Review Article

Trigeminal nerve palsy associated with intracranial aneurysms: Scoping review

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ABSTRACT

Background: Trigeminal nerve palsy (TNP) in patients with intracranial aneurysms (IAs) results from the disease process or its treatment. We systematically reviewed the literature on trigeminal palsy in patients with IAs.

Methods: PubMed, ScienceDirect and Web of Science were searched according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. Data extraction and quality assessment were performed according to preestablished guidelines.

Results: Twenty studies were included, yielding 69 patients with TNP and IAs. The mean age was 56.9 years and females accounted for 76%. Among the total cases, a cavernous internal carotid artery aneurysm was found in the vast majority (93%), followed by 7% of aneurysms in the basilar artery-superior cerebellar artery, posterior communicating artery, and anterior communicating artery. 96% of the aneurysms were classified as large to giant-sized. Out of the total number of cases, the majority (90%) exhibited trigeminal palsy at the time of their initial presentation. Only a small proportion ($n = 7$, 10%) developed fifth nerve palsy subsequent to the treatment of their aneurysms. Concurrent versus isolated TNP were exhibited in 79.7% and 20.3% of the cases, respectively. Finally, in terms of outcome, complete recovery from trigeminal palsy was achieved in 76.7% (26/34), with a duration of resolution of <6 months in 77.3% (17/22).

Conclusion: Trigeminal nerve palsies are correlated with IAs, and this correlation depends mainly on the location and size of the aneurysms.

Keywords: Aneurysm-induced trigeminal nerve palsy, Intracranial aneurysm, Trigeminal nerve palsy, Trigeminal neuropathy

INTRODUCTION

Cranial nerve palsy (CNP) can occur among individuals with cerebral aneurysms as an early clinical presentation or as an outcome of treatments. As a clinical finding, CNP might be part of a

symptom cluster in individuals with intracranial aneurysms (IAs).[24,34,37] The anatomical closeness provides the context for the relationship between a CNP and an aneurysm. Typical cases include oculomotor nerve palsy caused by a posterior communicating artery (PcomA) aneurysm.[23] Trigeminal nerve palsy (TNP) is typically presented as numbness in the skin or mucosal membranes in the distribution of the trigeminal branches and/or neuropathic weakness in the muscles of mastication.[37] These symptoms are collectively named trigeminal neuropathy. Several etiologies have been encountered as attributions to the TNP, including traumatic, vascular, inflammatory, demyelinating, infectious, and neoplastic disorders.[26] Among all these instances, the prevalence of TNP attributed to IAs is yet not reported. Potential mechanisms of TNP-IA association are mass effect, increased intracranial pressure due to aneurysmal rupture, or nerve damage during aneurysmal treatment.[24,34,37]

Many case series and reports dealing with the role of the fifth cranial nerve in IAs have been found in the literature. Nonetheless, the TNP has not been studied systematically in patients with nurtured IAs. In this systematic review, we comprehensively summarize the research on TNP as a result of IA, emphasizing aneurysmal and TNP features and discussing the potential mechanisms.

MATERIALS AND METHODS

Literature search

A systematic review was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses statement.[20] On October 8, 2023, systematic database investigations were undertaken, encompassing PubMed, ScienceDirect, and Web of Science. The devised search algorithm utilized Boolean conjunctions, specifically "OR" and "AND," incorporating a combination of the following keywords: ([trigeminal OR 5th cranial nerve OR CN-V] AND [palsy OR paresis OR injury OR dysfunction]) AND (IA). Postsearch, all acquired studies were integrated into Rayyan for deduplication and subsequent analysis.

Study selection

Inclusion and exclusion criteria were predetermined. Articles were selected if they included at least one patient manifesting TNP and diagnosed with an IA. TNP was defined as a clinical diagnosis denoting the dysfunction of the trigeminal nerve either at presentation or later due to the treatment, as recognized or reported by the respective authors. For IAs, the diagnosis should be based on computed tomography angiography, magnetic resonance angiography, or digital subtraction angiography. Inclusion was also for original data (not duplicated or referenced from other studies). The presence of other CNPs was deemed acceptable; however, traumatic CNPs were excluded. For aneurysms, any extracranial, bilateral, or multiple aneurysms unrelated to the course of the trigeminal nerve were excluded. Exclusions were also for studies with no relevant data on TNP or aneurysmal characteristics, non English language-based studies, conference abstracts, technical reports, book chapters, letters to the editor, editorials, radiological studies, anatomical studies, or those employing animal or cadaveric subjects. Although review articles were excluded from the review, references to these studies were screened to determine whether other original studies were relevant.

Four distinct reviewers systematically scrutinized the titles and abstracts of the retrieved articles, advancing to comprehensive text analysis for those aligning with the stipulated inclusion criteria. Discrepancies, when encountered, were mediated by a fifth reviewer (A.M.). Following the pre-established guidelines, relevant articles were integrated, and their reference lists were examined for additional pertinent studies.

Data extraction

Five distinct reviewers initiated the data extraction, which was subsequently cross-checked for accuracy by reviewer A.M. Missing data from the included studies were not reported by the authors. The data assessment parameters encompassed author - year, country of origin, study design, population size, age demographics, gender distribution, the status of aneurysm rupture with Hunt and Hess score and modified Fisher scale, aneurysm characteristics which include location, size, side and morphological type, trigeminal palsy characteristics which include timing of palsy, laterality, side, multiplicity, severity, description of trigeminal branches involvement, treatment method, outcome of trigeminal palsy, duration of resolution in months, follow-up duration in months, and modified Rankin scale. Other parameters, such as presentation description, potential mechanism of palsy, and the presence of daughter cysts, were also included.

Data synthesis, quality assessment, and statistical analyses

The primary outcome was centered on delineating the clinical characteristics of patients with TNP and IAs alongside their associated outcomes. Jamovi 2.4.1.0 was used for the descriptive analysis. Continuous variables are summarized as medians and ranges, while categorical variables are as frequencies and percentages. For each article, the level of evidence was independently evaluated by two reviewers (A.M. and H.A.) upon the 2011 Oxford Centre for Evidence-Based Medicine guidelines, and the risk of bias was assessed using the JBI checklists.[5,14]

RESULTS

Study selection

Figure 1 illustrates the literature screening. The preliminary search across PubMed, ScienceDirect, and Web of Science databases retrieved 159 articles, distributed as follows: PubMed (n = 88), ScienceDirect (n = 61), and Web of Science (n = 10). After a meticulous full-text review, 20 studies were deemed suitable for inclusion [Supplementary File 1].[1,31,4,7,9-13,15,18,22,25,29,32-35,38,40] Most studies were conducted in Japan (9, 45%) or in the United States (6, 30%). All incorporated studies are either case series or case reports. Quality assessment returned a low risk of bias for all studies [Supplementary File 2], predisposing this review to an overall low risk of bias.

General aneurysmal characteristics

A cohort of 69 patients was included in our study [Table 1]. The mean age was 56.9 years (range, 6–88). Out of 47 patients, the sex-based analysis revealed that females accounted for 76.6% (n = 43). The aneurysmal location was dominated by the cavernous internal carotid artery (ICA) in 92.75% of cases (n = 64), followed by basilar artery-superior cerebellar artery (BA-SCA) and PcomA (each 2.9%, n = 2), and a single case of anterior communicating artery (AcomA)

(1.45%). In terms of aneurysmal size, a cohort of 53 subjects was conducted. Remarkably, giant-sized aneurysms were exhibited in the majority of cases (n = 34, 64.15%), followed by large sized (n = 17, 32.1%). Only two cases, one of small sized and the other of medium sized (1.7%) were found in our review. Regarding rupture status, 94.2% (n = 65) of the cases had unruptured aneurysms, while only 5.8% (n = 4) of the aneurysms were ruptured. For the cases with ruptured aneurysms, the inclusion of the Hunt and Hess and modified Fisher scales were initially considered parameters. However, given the constraints of limited available patient data, we ultimately opted to exclude them from analysis. Among 17 cases studied for the aneurysmal side, right-sided and left-sided aneurysms were found in 12 (70.6%) and 5 (29.4%) of the cases, respectively. For morphological type, saccular aneurysms dominated in the vast majority of the cases (n = 67, 97.1%), leaving only two cases with fusiform type (2.9%). The study of treatment modality was conducted in the included studies. For the cases that did not mention their treatment, we consider it as conservative management. Consequently, among 68 subjects, the bypass was the most frequently used method at 44.1% (n = 30), followed by conservative management (33.8%, n = 23), flow diversion (10.3%, n = 7), and finally, clipping and coiling (each 5.9%, n = 4).
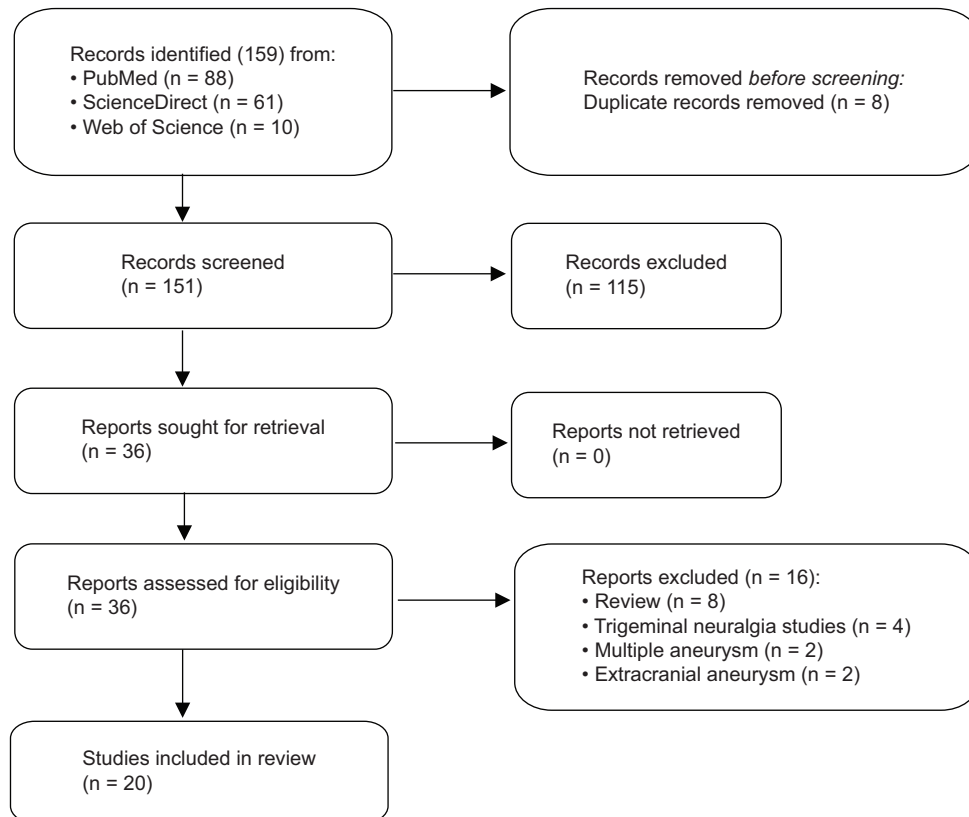

Figure 1: Flowchart of the study selection process.

Table 1: Summary of the included studies.

Characteristics	Value (%)
General characteristics	
Cohort size (no.)	69
Demographics	
Age (years), median (range)	56.9 (6–88)
Gender (female) (*n*=47)	36 (76.6)
Aneurysm characteristics	
Aneurysm location (*n*=69)	
ICA-cav	64 (92.75)
PcomA	2 (2.9)
BA-SCA	2 (2.9)
AcomA	1 (1.45)
Aneurysm size (*n*=53)	
Small (0–4.9 mm)	1 (1.89)
Medium (5–9.9 mm)	1 (1.89)
Large (10–24.9 mm)	17 (32.1)
Giant (>25 mm)	34 (64.15)
Aneurysm side (*n*=17)	
Right	12 (70.6)
Left	5 (29.4)
Rupture status of aneurysms (*n*=69)	
Unruptured	65 (94.2)
Ruptured	4 (5.8)
Morphological type of aneurysms (*n*=69)	
Saccular	67 (97.1)
Fusiform	2 (2.9)
Treatment type (*n*=68)	
Clipping	4 (5.9)
Coiling	4 (5.9)
Bypass	30 (44.1)
Flow diversion	7 (10.3)
Conservative	23 (33.8)
TNP characteristics	
Timing of TNP (*n*=69)	
Pretreatment	62 (89.86)
Posttreatment	7 (10.14)
Laterality of TNP (*n*=69)	
Ipsilateral	66 (95.7)
Contralateral	1 (1.45)
Bilateral	2 (2.9)
The multiplicity of TNP involvement (*n*=69)	
Yes	55 (79.71)
No	14 (20.29)
Frequency of cranial nerve involvement (*n*=169)	
Oculomotor	55 (32.5)

(Contd...)

Table 1: (*Continued*).

Characteristics	Value (%)
Trochlear	52 (30.8)
Abducent	41 (24.2)
Optic	20 (11.8)
Facial	1 (0.6)
Severity of TNP (*n*=69)	
V1	8 (11.59)
V1-V2	11 (15.94)
V2-V3	4 (5.8)
V1-V3	44 (63.77)
Motor+Sensory	2 (2.9)
TNP outcome (*n*=34)	
Complete recovery	26 (76.47)
Partial recovery	8 (23.53)
Duration of resolution (*n*=22)	
<6 months	17 (77.27)
More than 6 months	5 (22.72)

AcomA: Anterior communicating artery, BA-SCA: Basilar artery-superior cerebellar artery, TNP: Trigeminal nerve palsy, ICA-cav: Internal carotid artery-cavernous portion, PcomA: Posterior communicating artery

TNP characteristics

Within the scope of our study, we aimed to ascertain the temporal correlation between TNP and aneurysmal treatment. The majority of cases had TNP at the presentation (*n* = 62, 89.9%). Only 7 cases (10.1%) had TNP after the treatment of aneurysm. In the study, TNP laterality was examined among the included cases. Studies that did not specify the side of TNP cases were considered ipsilateral. Consequently, 66 (95.7%) exhibited ipsilateral TNP, 2 (2.9%) bilateral TNP, and a single case (1.45%) contralateral TNP. For the multiplicity of TNP involvement, 55 (79.7%) had CNP other than trigeminal palsy, and the remaining 14 (20.3%) had isolated TNP. Further analysis of the former group revealed that oculomotor CNP was the nerve dysfunction most commonly associated with TNP, followed by trochlear and abducent (combined 87.5%). Concerning the severity of TNP, the majority of cases exhibited sensory involvement in the branches V1-V3 (*n* = 44, 63.8%), with V1-V2 branches following suit (*n* = 11, 15.9%) [Table 1]. In terms of TNP outcome, a cohort of 34 patients was conducted in the study. Complete functional recovery from TNP was identified in 26 cases (76.5%) and partial recovery in 8 (23.5%). The duration of TNP resolution among 22 subjects was mostly <6 months (*n* = 17, 77.27%).

DISCUSSION

Anatomical association of TNP and IA

The trigeminal nerve is the largest and most complex of the cranial nerves. It serves as a major conduit of sensory input from the face and provides motor innervation to the muscles of mastication. Its course from origin to insertion is divided into the brainstem, cisternal segment, Meckel's cave segment, trigeminal ganglion, and peripheral divisions (ophthalmic, maxillary, and mandibular divisions).[6] Throughout its path, the trigeminal nerve maintains anatomical relations with vital neurovascular structures, notably the SCA; anterior inferior cerebellar artery; cranial nerves III, IV, and VI; and cavernous ICA. Its complex intracranial course and neurovascular relationships render it vulnerable to injury during surgical procedures or from various compressive vascular pathologies. A spectrum of pathophysiological processes may affect the trigeminal nerve, including traumatic, vascular, inflammatory, demyelinating, infectious, and neoplastic disorders.[26] Among all these instances, the prevalence of TNP attributed to IA is not reported.

Our objective in this systematic review was to encompass all instances of trigeminal palsy attributed to IAs, whether they occurred before or following treatment, and discuss the potential mechanisms that lie behind it.

Intracranial aneurysmal characteristics

The location of the IAs in patients with TNP was examined to determine its significance. In our review, cavernous ICA aneurysm was found in the vast majority of TNP cases (93%), followed by BA-SCA and PcomA (about 6%). It seems evident that the anatomical proximity of these aneurysms to the course of the trigeminal nerve plays a major role in the causation of the TNP, as the mass effect exerted by them is a relatively common etiology for CNP at presentation time.[3,27,28] In support of that, a combination of large to giant-size aneurysms was found mostly in the literature (96%). One can propound that the larger size of the aneurysm can be one of the most important reasons for direct compression on the trigeminal nerve and damaging its neurons. In parallel to our finding, Durner *et al.* found that CNP was significantly more frequent in giant-sized cavernous ICA aneurysms.[2]

The underlying mechanism of CNP in unruptured aneurysms is distinct from that observed in ruptured aneurysms. Various prospective explanations for the pathogenesis of this correlation have been put forth in the literature. As described previously, in the case of an unruptured IA, the cranial nerve may experience direct pressure from the expanding aneurysmal sac or nerve ischemia.[30] Conversely, the impact on cranial nerves in cases of ruptured IA may be ascribed to either direct trauma resulting from the blood flow, the formation of a localized hematoma, or irritation induced by the blood's presence in the subarachnoid space.[8,16,21,39] The analysis revealed that approximately 94% of the TNP cases were associated with unruptured aneurysms, providing further support for the aforementioned concept of direct compression.

A norm, nevertheless, is subjected to exceptions. Thorat and Hwang discovered a noteworthy case involving TNP and a ruptured small AcomA aneurysm.[33] Although the trigeminal nerve of this patient may be injured from the resulting subarachnoid hemorrhage, it appears implausible that direct compression could induce TNP in such an aneurysm, given its size and location. Nevertheless, trigeminal palsy in this patient was observed subsequent to aneurysm treatment, indicating that it is a complication that may arise after the procedure. The bottom line is that the role of IAs in inducing TNP extends beyond the aneurysmal sac itself or blood leakage; it also encompasses the management of these conditions, whether it is during the surgical procedure itself or in the days that follow. Our review identified seven cases (10%) that exhibited posttreatment TNP; all of these cases underwent endovascular treatment for their IA, representing about half of the total endovascular cases found in our analysis. Despite the small number of cases, the TNP in these patients may be ascribed to the aneurysmal increase in size in the follow-up,[19,36] taking us back again to the aforementioned theory of direct compression.

Trigeminal palsy characteristics

Studying the features of TNP revealed that most of these cases exhibited ipsilateral TNP and had complete sensory involvement in the branches V1-V3. Partial involvement of the trigeminal nerve branches was found in scattered cases, and only two cases exhibited dysfunction of the motor component of the trigeminal nerve. In addition, the involvement of other cranial nerves was also dominant, notably the oculomotor, trochlear, and abducent nerves. The presence of these nerves in a confined space renders them vulnerable to mass compression by adjacent arterial aneurysms, especially cavernous ICA aneurysms, which were seen in the majority of the included cases. In our analysis, the concurrent TNP was seen in about 80% of the studied cases. Most of these subjects had large to giant-sized cavernous ICA aneurysms. Multiple combinations of cranial nerve palsies attributed to cavernous ICA aneurysms have been reported in the literature.[1,4,12,17,18] The remaining 20% of cases had isolated TNP; most of these cases harbored small to medium-sized aneurysms or BA-SCA aneurysms, which may compress the trigeminal nerve in close proximity to its insertion, distinct from the other cranial nerves.

Notwithstanding the restricted number of studied cases, the analysis of long-term recovery from TNP unveiled a commendable recovery rate, estimated at approximately 100% of partial to complete recovery, within a timeframe

of <6 months. This holds significant clinical relevance during the initial consultation with the patient, as it aids in elucidating the prognosis and potential for long-term recovery of their trigeminal palsy.

In brief, the initial analysis of the correlation between TNP and IA revealed that the most frequently documented causative aneurysms of TNP in the literature are unruptured, large to giant-sized cavernous ICA aneurysms, mostly in the pretreatment state, ipsilateral, and concurrent with other CNPs. Finally, most cases exhibited a favorable outcome, with a majority achieving complete resolution of their TNPs.

Limitations

Our study has certain limitations. Diagnosing TNP is challenging, relying heavily on clinical confirmation. Most reviewed articles were retrospective, introducing inherent selection bias due to preexisting data. Although we found no evidence of publication bias, institutional reporting bias cannot be entirely ruled out. Our focus on English-language articles may have excluded relevant research in other languages, possibly introducing language bias. In addition, the majority of included studies were cohort analyses and case reports, which inherently emphasize unique clinical scenarios, potentially deviating from broader patient population characteristics.

Despite these limitations, our review offers valuable insights into the TNP-IA relationship, providing a comprehensive overview of the literature and highlighting areas for future research.

CONCLUSION

Trigeminal nerve palsies are correlated with IAs, and this correlation depends on the location and size of the aneurysms, typically leading to ipsilateral complete facial numbness at the presentation, usually with the involvement of other cranial nerves.

Ethical approval: Institutional Review Board approval is not required.
Declaration of patient consent: Patient's consent is not required as there are no patients in this study.
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Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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